Understanding Genetic Anomalies Resulting from Inbreeding, Their Origins and How to Identify Anomalies Caused by Such; Implications for Cancer Research

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Simon Edwards
Research Acceleration Initiative

Introduction

A oft-repeated explanation for the causes of genetic anomalies owed to inbreeding is that these defects are somehow the result of a lack of an alternative choice of genes when a gene is "bad." If either parent had so many bad genes, the phenotype associated with such corruption would be evident in the parent(s.) This trope has been repeated since a time shortly after the discovery of the structure of DNA despite never amounting to more than pure speculation. As a result of this trope, however, little research has been done into the exact mechanism through which inbreeding-related copy errors within zygotes come about, most likely due to a false assumption that the science is settled.

Abstract

During the formation of a zygote, in the very earliest stage of development (in which the genetic code of the zygote is determined by randomly selecting genes from one parent or the other,) in any ordinary fertilization regardless of whether inbreeding is a factor, there is a chance that the available base pairs from each parent will match. A choice must be made through a molecular process as to whether to derive a base pair from either one parent or the other. In the case that, for instance, a double-cytosine (CC) is made available from both parents at a particular "position," the molecular scissors responsible for selecting either one parent's genes or the other may not be able to copy either due to as-yet undiscovered specificity of function of those CRISPR molecules to adenine, cytosine, thymine, or guanine. These molecular scissors are agnostic to the sex of the parent that they draw DNA from, despite the belief of many to the contrary, but are instead programmed to copy only specific base pair chemicals. The diffusion an assortment of these "scissor" molecules in the zygotic medium determines which base pairs are selected from which parent.

If, however, for instance, a molecule is primed (much like an antibody) to want to copy adenine rather than cytosine and it finds itself between two sets of double-cytosines, it would not be able to perform its function. If a sufficient length of time elapses with that segment of the zygote's genetic code not having been selected from either one parent or the other, the zygote's own internal DNA replication process fills in the gap by making an inappropriate copy of neighboring regions of its own, newly established genetic code.

When only, perhaps, less than two dozen consecutive base pairs are identical, this is unlikely to create a problem given that other "scissor" molecules are in the

region and can pick up the slack of the molecules rendered inactive by their own inability to cut and paste a cytosine. In the case of inbreeding, however, there are hundreds of consecutive identical base pairs, a fact that forces large gaps to be filled in by the new lifeform's own internally generated CRISPR molecules. These molecules know only that "something" is supposed to fill in the void left by the inability of the primary CRISPR process (driven by the reproductive cells of the parents) to be completed. This is analogous to one painter finishing a portrait started by another and rather than intelligently extrapolating the intent of the first painter, instead makes a series of exact copies of the upper-left quadrant of the portrait, resulting in something that does not resemble a portrait per se.

When an exact copy is made of a neighboring zone, it might be identified in genetic testing by analyzing the level of tension with which the DNA is coiled. These improperly duplicated regions would, I would suggest, tend to be rendered permanently inactive or perhaps underactive, meaning that their DNA would be coiled more tightly around their substrate.

Conclusion

Developing a methodology for identifying which genes in an individual genetic code were set by this as-yet-unnamed "fill-in-the-gaps" process may enable researchers to focus their attentions on those regions i.e. they are more likely to cause problems. Understanding the nature of this secondary copying process of last resort may also provide insights into the nature of the self-replication processes that drive cancers.